but not excessive; the thirst was not noticeably increased, and nothing else abnormal was discovered. The child was placed on an exclusively meat diet, with milk, eggs, and red wine, and by the end of April there was only one per cent. of sugar in the urine, and by the middle of May one-fourth per cent. From this time the child improved in strength, and was allowed to have a mixed diet, and by the middle of June the urine was free from both sugar and albumen, and the patient soon became as strong and well as ever.

This case is of unusual interest, both from the rarity of its occurrence as a sequela of scarlet fever and from its unusually favourable result. Külz, in his article on diabetes mellitus in Gerhardt's *Handbuch der Kinderkrankheiten*, states that out of 111 cases of this disease only seven recovered. The same author mentions that the cause in two cases appeared to be measles, but no case is attributed to scarlet fever. Redon, in his collection of cases of diabetes,[1] gives, among other causes, weakness from previous diseases, such as measles, scarlet fever, typhoid, etc. Yet on looking over the original articles from which he gathers his cases scarlet fever in no instance is found to be a cause. Thomas, in Ziemssen's *Handbuch der Allgemeinen Pathologie*, Band i., s. 290, speaks of the appearance of sugar in the urine with cerebral symptoms occurring during the stage of fever in scarlet fever, but not as a sequela of that disease. In view of this case of Zinn's it would be well during a convalescence from scarlet fever to examine the urine carefully for sugar as well as albumen where the patient does not gain in general strength as fast as he ought to.—*Boston Med. and Surg. Journ.*, July 26, 1883.

Acetonuria and Diabetic Coma.

The termination of diabetes by acetonuria or acetone intoxication, if one may judge by recent statistics, seems to be much more frequent than is generally supposed. S. MACKENZIE has noted it 19 times in 37 cases of diabetes, and 26 times in 43 other cases observed in Guy's Hospital. From these facts he believes that he is justified in concluding that young diabetics frequently die in coma, the disease developing in these with a certain acuity, and the lungs being sound or invaded only by the lesions of commencing phthisis.

In a work on diabetic coma Frerichs reports no less than 25 cases coming under his own observation, of which he made detailed observations. He divided these into three groups: the first comprehending cases of diabetes with rapid death, the patients having died in a comatose state in a few hours after being seized, without prodromal symptoms, with general feebleness, failing pulse, and cold extremities. Some of the patients in this first group already had marasmus. In the second group the comatose state was preceded by prodromal symptoms, general feebleness, gastric symptoms, nausea, vomiting, and obstinate constipation, or by some local affection such as dental abscess, pharyngitis, phlegmon with tendency to gangrene, bronchitis, or broncho-pneumonia. Before falling into the somnolent and comatose state the patients have headache, agitation with delirium, great pain, sometimes true accessions of mania, and dyspnœa; at times they make deep inspirations and expirations; the pulse becomes rapid and feeble; the temperature descends below the physiological level. This state lasts from three to five days. In the third group Frerichs places the diabetic patients who, without the least dyspnœa or pain, with firm pulse and well preserved vital forces, are suddenly taken with headache, a kind of intoxication, and finally somnolence, coma, and death.

The numerous remedies which have been proposed against these accidents seem to be equally inefficacious. Frerichs cites, among others, transfusion, the administration of oxygenated water, stimulants, weak solutions of phosphate and chloride of sodium, subcutaneous injections of ether, camphorated oil, antizymotics, carbolic acid, salicylic acid, etc. Of the causes of diabetic coma we are still ignorant; the alterations of the nerve centres, as offered in explanation by some, are not constant; others speak of thickening of the blood by an accumulation of sugar in it, a morphological and functional alteration of the red globules. This gratuitous hypothesis has led to dangerous therapeutic measures; others have attempted to unite diabetic coma and uræmia by ascribing the former to insufficient excretion of urine. Frerichs relates a case of uræmia in a diabetic which conclusively proves that there can be no possible assimilation between the intoxication of Bright's disease and that of diabetes which produces fatal coma. Ebstein has attributed diabetic coma to a retention of the excrementitious products of the blood, occasioned by a necrosis of the renal epithelium at the level of Henle's tubes. This lesion is too inconstant to explain the result. Frerichs has observed that in diabetics the epithelial cells of the tubules were constantly the seat of a hyaline degeneration, due to an accumulation of glycogen in the cells. The sole fact of its constancy in diabetics, who have succumbed to it matters not what complication of this degeneration, cannot be held to be the cause of the comatose condition.

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[1] Virchow und Hirsch, Jahrb., 1877, Band ii.

Frerichs has never been able to discover traces of fat emboli in the pulmonary vessels of the glomeruli of the kidney, of the liver or brain, as has been suggested as a possible fact in the pathogenesis of diabetic coma. In fine, says Ricklin, it is commonly admitted to-day that acetone and diacetic ether, which for a long time have been considered as causes of diabetic intoxication and coma, have no part in its development; diacetic ether pre-exists neither in the urine nor in the blood, and both acetone and diacetic ether are frequently injected into the veins of animals in laboratories.

Jaksch has shown (*Zeitschr. f. Physiol. Chem.*, t. vi. 541, 1882) that acetone is a product of malassimilation which is found in the blood and urine in the normal state, and that it is increased in certain pathological sates; he says that every febrile affection, from whatsoever cause, is accompanied by acetonuria; and that the quantity of acetone eliminated by the urine, which may reach five centigrams in twenty-four hours, is in direct proportion to the intensity of the fever. Jaksch has seen cases of diabetes in which only normal quantities of acetone were eliminated by the urine; other cases present a true acetonuria, the urine turning red when heated with perchloride of iron. In still others, advanced diabetics, this reaction coincided with the presence of a considerable quantity of acetone in the urine. Of this third class many patients succumb to diabetic coma. Jaksch has twice found pronounced acetonuria in young men suffering with gastric troubles, saburral tongue, anorexia, constipation alternating with diarrhœa, cephalalgia, slight tumefaction of the spleen without fever. In these cases the urine heated with perchloride of iron gave the red color. He has also found a large quantity of acetone in the urine of persons with hydrophobia, as well as in cases of cancer of the stomach, in a case of cancer of the œsophagus, and in one of cancer of the stomach and pancreas. In another case dying in a state precisely similar to diabetic coma, the autopsy showed cancer of the pylorus, with metastatic foci in a large number of organs. (Ueber Pathol. Acetonuria, *Zeits. für Klin. Med.*, t. v. fasc. iii. p. 346.)—*Gaz. Méd. de Paris*, July 7, 1883.

Resorcin in Whooping Cough.

In an interesting article, recently published, Dr. MONCORVO, Professor of Diseases of Children in the Polyclinic of Rio de Janiero, strongly advocates the employment of resorcin in pertussis.